CONCLUSIONS. From our investigations we must conclude that the serum diagnosis of tuberculosis as used by Arloing and Courmont is not a specific sign of the presence of clinical tuberculosis, since healthy and tuberculous serums have practically the same agglutinating property. We therefore conclude that the serum diagnosis of tuberculosis, and especially for the early diagnosis of tuberculosis, is of no value.

# THE CLINICAL, ANATOMICAL, AND PATHOLOGICAL COMPARISON OF TUBERCULOUS CAVITIES IN THE LUNGS.[1]

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THIS paper has been prepared with the view of reconciling some of the divergent views in regard to cavities in pulmonary tuberculosis, particularly from the standpoint of diagnosis. From its inception, it has been the custom at the Phipps Institute to bring before the members of the staff at its weekly meetings the results of each autopsy. Black-board diagrams of the clinical findings are made and these are then compared with the fresh anatomical specimens.

The 53 cases I have analyzed resulted in death during the year 1905. The autopsies were made by C. Y. White, pathologist to the Phipps Institute, and the clinical examinations by members of the house staff, numbering eight.

The opportunity which a large number of autopsies has afforded of verifying clinical findings has shown that errors are not infrequent. Thus, a cavity is diagnosed as being present which the postmortem fails to reveal, or a cavity actually present has escaped notice. The question of whether a lung contains a cavity is of considerable importance from the standpoint of prognosis. Small or fairly large cavities which are sharply defined, and especially if dry, make for a fairly favorable prognosis, while communicating cavities or single cavities with active constitutional symptoms render the prognosis grave. In any case, however, the prognosis should be guarded, owing to the possibility of a sudden and fatal hemorrhage. Quite recently I saw a man whose history indicated disease of some years' standing and who had in the apex of the right lung a large, quiescent cavity. He had a slight cough and some expectoration in the morning, but no other symptoms. A favorable prognosis was given, but he died suddenly one week later from a large pulmonary hemorrhage.

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[1] Read at the meeting of the National Association for the Study and Prevention of Tuberculosis, Washington, D. C., May 17 and 18, 1906.

The possibility of such an accident can never be guarded against, as it is well known that once an excavation has formed it is never obliterated. Furthermore, there is no means of knowing whether the bloodvessels have become completely thrombosed. Another menace to the individual with a cavity is the constant danger of mixed infection. Even under the most favorable circumstances these excavations are points of least resistance, and no lung containing them is as safe from outside influences as the one in which the infected area is firmly walled off by fibrous tissue. On the other hand, it sometimes happens that the patient's condition is improved by the formation of a cavity, for with the removal of a necrosed area an immediate improvement is noted in the constitutional symptoms. Such an individual, however, must always be subjected to the dangers mentioned above.

Perhaps, the most striking feature of tuberculosis of the lungs is the excavations which occur. In the chronic types this feature is present in the majority of cases. In the acute forms of the disease, in the so-called pneumonic and bronchopneumonic types, excavations are not unusual, particularly in the bronchopneumonic form, although the process may be so acute as to cause death before breaking down of the caseous area occurs. This is the type of the disease usually occurring in children and the majority of the lower animals. Even in general miliary tuberculosis excavations are not uncommon and in some instances must be looked upon as the infecting source of the generalized process.

I have heard it asserted that every case of tuberculous infection of the lungs develops some evidence of excavation, however small. Of 78 cases which Flint analyzed from this standpoint 62 showed cavitation; of the 16 without cavities, 5 were of the acute miliary type, leaving 10 of the more chronic forms in which no excavations had occurred. Of 53 cases examined at the Phipps Institute during 1905, 50 had well-marked cavities. The longer the tuberculous process lasts, no matter how slow its progress, the more certain is excavation to occur, although in very latent and chronic cases, so-called fibrous phthisis, the disease may last for years and at death show no evidence of cavity formation. In those types in which the tuberculous process, aided by mixed infection, pursues a more or less rapid course, excavations occur quickly. Cavity formation is usually a gradual process, at least so far as we are able to judge by physical signs. Occasionally, however, the excavation is formed suddenly, and in an area where consolidation had been noted for weeks the unmistakable signs of cavity may be detected.

The wall of a cavity consists of three parts: (1) A caseous lining in which all trace of lung tissue is lost and the margin of which toward the cavity is breaking down; (2) a zone in which early tuberculous changes, both interstitial and alveolar, are seen; and (3) an outside zone of small-celled infiltration (West). Similar changes may be going on in adjacent caseous areas with communicating fistulous tracts, giving the lung a honey-combed appearance. Recently formed cavities have an irregular outline, are ill defined, the interior is ragged, and there is an absence of a distinct wall. Cavities of this description are met with in the more or less acute tuberculosis and in the chronic types in those portions of the lung where the disease is advancing. When the process is less acute, or has remained localized for some time, the outer zone of small-celled infiltration develops into more or less well formed fibrous tissue. When this occurs the caseous lining is less, as is also the area of tuberculous infiltration.

Cavities which have existed for a long time develop a thick fibrous surrounding and the caseous lining gradually changes into granulation tissue, which is in turn replaced by fibrous tissue, giving the interior a smooth lining. Secretion then ceases or nearly so, and the cavity is designated dry or quiescent. Various stages of this process are met with in chronic tuberculosis.

Cavities of any considerable size contain in their walls or in the trabeculæ transversing them, bronchi and bloodvessels. The latter in the chronic cases usually become thrombosed and obliterated, although even in these cases a fatal termination may be brought about by rupture of a small aneurysmal dilatation. In the destruction of tissue a number of bronchi are inevitably involved. As a rule, most of these bronchi are destroyed by the tuberculous process or occluded by the fibrous tissue. External communication is then brought about by the breaking down of the tuberculous process or by ulceration of the surrounding tissue, until a large bronchus with its thicker walls is incorporated in the diseased area. This being more resistant to the necrotic process, remains patulous. It is not, however, often that a cavity is drained by more than one or two such openings.

When a number of such areas remain patent or when the opening is single and small, the physical signs are imperfect; to this fact must be ascribed the failure in some instances to recognize excavation.

According to West: "In the more acute cases there will be a larger amount of caseous material, more bacilli as a rule, and a greater likelihood of fragments of lung tissue. In the more chronic cases the amount of pus is larger, and it is observed in the sputum that the amount of pus and the number of bacilli often stand in inverse proportion to each other."

To Louis we are indebted for the observation that when tuberculosis attacks the lungs it almost invariably involves the apices first. This fact was further elaborated by Kingston Fowler, who showed that it is not the extreme apex which is involved, but a point an inch to an inch and a half below the apex, and that after tending to spread backward the course of the disease is downward, the upper lobe and apex of the lower lobe on the primary side being involved before the apex of the opposite side is attacked.

As excavation is the final stage in the tuberculous process, it is but natural to look for the evidence of cavity formation where the disease first appeared. In an analysis made by Ewart cavitation occured at the apices, 282 times; the dorso-axillary region, 227 times; the mammary region, 189 times; the sternal region, 61 times; the base, 32 times.

The distribution in the 50 cases at the Phipps Institute was as follows: right upper lobe, 15 times; left upper lobe, 6 times; right middle lobe, 1 time; right lower lobe, none; left lower and both upper lobes, 1 time; left and right upper lobes, 30 times; giving a total of 76 cavities.

In this series there was no instance of a primary basic cavity. This condition is rare, occurring in less than 1 per cent. of cases. Basic cavities in association with apical disease, however, are not uncommon, particularly when the disease is advancing rapidly.

The size of the cavities ranged from that of a cherry (1 x 1 cm.) to involvement of nearly an entire lung. In estimating the number of cavities present, small communicating cavities have been considered as a single large excavation, recognizing, however, that communicating small cavities may originate separately and coalesce from ulceration of the intervening lung tissue, or they may be separate points of softening in a large caseous area. Clinically the effort was made to distinguish this type.

It is evident that a pulmonary cavity at the apex is almost certainly tuberculous, and with the presence of the bacilli is invariably so. In most cases of chronic tuberculosis there is a slight degree of dilatation and thickening of the bronchial walls, but it rarely happens that these dilatations are sufficiently large to give physical signs. In two of our cases cavity formation and marked bronchiectasis at the apex co-existed, giving the signs of a single large cavity.

Involvement of the pleura over cavities is very common. Dense pleural thickening occurred in 60 of 76 cavities we examined. In the chronic forms this thickening is almost constant. In acute forms of the disease and in the advancing areas of the chronic cases when the breaking down is rapid, ulceration through the pleura with a resulting pneumothorax is not unusual. I have met with several instances in which the pleura had been perforated by an ulceration, but the dense adhesions presented collapse of the lung. Rarely a cavity may ulcerate through the chest walls. In one case which I have seen this accident was prevented by a rib which had become markedly necrosed.

In comparing the clinical with the pathological findings in these 50 cases, it was found that of the 76 cavities present, 58 had been recognized, while 18 had escaped notice. The majority of the cavities not recognized were very small; three of them, however, had attained the size of a lemon (4 x 5 cm.).

It is usually stated in the text-books that a cavity must have attained the size of an English walnut before it gives signs sufficient for its recognition. The situation of the cavity, however, has a more important bearing on its recognition than its size. If superficially placed just beneath the pleura, cavities the size of a cherry (1 x 1 cm.) will often give well-marked signs, while much larger cavities deeply situated with a thickened pleura and normal and infiltrated lung tissue about them, will escape detection.

In the 58 cavities which were correctly diagnosed, lack of expansion, retraction of the apex, and flattening of the chest wall anteriorly were the rule. Bulging or fulness of the chest wall over the site of the cavity was not met with, although I have seen this phenomenon once.

In those cases in which it could be elicited the tactile fremitus was exaggerated in 34 instances, and diminished in 13.

The percussion note had a tympanitic quality in 36; was dull in 16; hyperresonant in 3; and in 3 instances was of the cracked pot variety of tympany.

The breath sounds were noted as cavernous in 29; amphoric in 19; bronchial in 13; and distant and indefinite in 7.

There was an absence of rales in 9. When present they were noted as being fine and moist in 16; consonating in 17; and gurgling in 16.

Vocal resonance was exaggerated in 51 instances, and diminished in but 1. Whispering pectoriloquy was present in 53 instances and absent 5.

In 8 instances cavities were correctly diagnosed as being communicating; this was about one-fourth of those actually of that character. The diagnosis was based upon the breath and voice sounds having several points of maximum intensity, in conjunction with a large area of coarse bubbling rales with a metallic quality, and symptoms indicating a rapid destruction of tissue.

The physical signs over those cavities (18) which were not recognized were almost invariably dulness, bronchial breathing, increase in vocal and tactile fremitus, and absence of whispering pectoriloquy. Most of these cavities were found postmortem to contain thick, creamy pus. Failure in the others may have been due to a number of small openings rather than to one or two large ones. On this point, however, there are no notes.

In the 10 instances in which cavities were believed to be present, but which at postmortem were not found, consolidated tissue around a bronchus was the source of error in 5. In 1 there was no adequate explanation. In 4 the error was probably due to transmission of the amphoric breathing and whispering pectoriloquy from a cavity on the opposite side. In these 4 instances the supposed cavities gave signs posteriorly only. Cavities as a rule give more marked signs anteriorly. Thus, of the 58 under consideration, the signs were present anteriorly, or anteriorly and posteriorly in 55 instances, while but 3 gave signs posteriorly alone.

From this analysis it is evident that our results were not as accurate as they might have been, particularly in the estimation of conditions simulating cavity formation.

As far as the recognition of cavities actually present was concerned the results were fairly good, as the majority of those escaping notice were very small and the conditions found postmortem, in a measure, explained the cause of failure.

Basing the value of the various signs on the frequency with which each occurred, the following results were obtained: whispering pectoriloquy, 55 out of 58; tympany, 39 out of 58; cavernous or amphoric breathing, 38 out of 58; gurgling or consonating rales, 33 out of 58. While whispering pectoriloquy was most frequently met with its value is somewhat impaired by its absence in cases of well-marked cavitation, and also by the fact that its presence occasionally leads to a diagnosis of cavity when other pathological conditions, notably consolidation about a bronchus, are the real causes.

In conclusion, it may be said that any one of the above signs is suggestive of a cavity and that the probability of its existence is increased by the combination of any two or more of them.

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# STATISTICS OF DIET IN SANATORIA FOR CONSUMPTIVES.[1]

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Hitherto, the chief weapons used in fighting tuberculosis have been fresh air and rest. But in such a desperate combat, every available weapon should be used—regularly graduated exercise, massage, hydrotherapy, mental diversion and suggestion, and last but not least, diet. Diet, however, while doubtless one of the most important agencies for the restoration of the consumptive's health, is also probably the most obscure and difficult of application.

The theories of diet in relation to tuberculosis are numerous and conflicting. Some authorities still cling to whiskey; most advocate meat, but there are a few whose experience has led them to discard meat; some emphasize the importance of cooking meat thoroughly, others maintain that it should be taken raw; eggs are recommended by

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[1] Read at the meeting of the National Association for the Study and Prevention of Tuberculosis, Washington, D. C., May 17 and 18, 1906.